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ORIGINAL ARTICLE

Association between Obesity and Metabolic Abnormalities in Women with Polycystic ovary syndrome

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ABSTRACT

Background: *Polycystic ovary syndrome (PCOS) is a common endocrine disorder of reproductive-aged women and is closely linked with insulin resistance and metabolic disturbances. Obesity amplifies these abnormalities, yet data on how obesity relates to specific metabolic abnormalities in South Asian women with PCOS remain limited. Asian-specific anthropometric thresholds are needed to classify risk accurately. Local evidence can guide screening and counseling in Bangladeshi clinical practice.* ***Objective:*** *To examine the association between obesity and metabolic abnormalities in women with PCOS.* ***Methods & Materials:*** *This cross-sectional analytical study was conducted at Popular Diagnostic Centre Ltd., Chattogram Branch, Bangladesh, from January to December 2021 and included 150 women with PCOS diagnosed by the Rotterdam criteria. Obesity was defined as BMI ≥25 kg/m^2 and abdominal obesity as waist circumference ≥80 cm. Metabolic syndrome followed International Diabetes Federation criteria. Comparisons and binary logistic regression were performed using SPSS version 25.0.* ***Results:*** *The mean age was 24.8 ± 5.1 years; 43.3% were obese, 64.7% had abdominal obesity and 52.0% had metabolic syndrome. Obese women had higher waist circumference, blood pressure, fasting insulin, HOMA-IR and triglycerides and lower HDL-C (p<0.05). Metabolic syndrome (70.8% vs 37.6%), insulin resistance (72.3% vs 37.6%) and dyslipidemia (96.9% vs 68.2%) were more prevalent in obese women (p<0.001). Abdominal obesity was associated with insulin resistance (adjusted OR 3.86), hypertriglyceridemia (3.42) and low HDL-C (2.71), but not impaired fasting glucose.* ***Conclusion:*** *Obesity, particularly abdominal obesity, is associated with insulin resistance, dyslipidemia and metabolic syndrome in women with PCOS. Routine metabolic screening and weight management should be offered to all affected women.*

Keywords: *Polycystic ovary syndrome; Obesity; Metabolic syndrome; Insulin resistance; Dyslipidemia.*

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrine disorder of reproductive-aged women, affecting an estimated 5 to 20% worldwide and insulin resistance with compensatory hyperinsulinemia is evident in the vast majority of affected individuals [1]. Diagnosis rests on the Rotterdam criteria, which require two of three features, namely hyperandrogenism, oligo-anovulation and polycystic ovaries, after exclusion of related disorders; insulin resistance and obesity are common but are not diagnostic criteria [2]. Beyond reproductive disturbance, the syndrome carries long-term metabolic and cardiovascular consequences and an international evidence-based guideline has been developed to standardize its assessment and management [3,4].

The interplay between insulin and androgens is central to its pathophysiology. Ovarian insulin resistance leaves steroidogenic activity intact, favoring hyperandrogenemia and androgens may in turn worsen insulin resistance, sustaining a self-perpetuating cycle [5]. Obesity acts on top of this background. Women with PCOS have a higher prevalence of overweight, obesity and central obesity than women without the syndrome and excess adiposity worsens most metabolic and reproductive outcomes, with central obesity linked to higher fasting insulin [6,7]. Obesity raises insulin resistance and inflammatory adipokine release, although it is neither necessary nor sufficient for the phenotype and its contribution differs across ethnic groups [8]. Clamp studies show that insulin sensitivity is reduced in PCOS independent of BMI, with higher BMI deepening the deficit [9,10].

The metabolic syndrome illustrates how these factors converge. A meta-analysis found that women with PCOS had more than threefold higher odds of metabolic syndrome, with

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the excess seen in overweight or obese women but not in lean women and with prevalence tracking obesity and metabolic features rather than hyperandrogenism [11]. Pooled prevalence estimates nevertheless vary widely with the diagnostic definition, from 7.1% to 37.5% [12]. A similar pattern is seen for dysglycemia, as ethnicity and obesity both contribute to the prevalence of impaired glucose tolerance and type 2 diabetes in PCOS[13] and a population-based cohort showed that type 2 diabetes risk was raised in overweight and obese women with PCOS but not in those of normal weight [14].

Most of this evidence originates from Western or mixed populations. Differences in body composition, diagnostic criteria and ethnicity make direct extrapolation to South Asian women uncertain. South Asian women are classified using lower, Asian-specific BMI and waist thresholds, so the relationship between obesity and individual metabolic abnormalities may differ in this group. In Bangladesh, published data on PCOS remain limited and evidence linking obesity status and abdominal obesity to specific metabolic abnormalities after age adjustment is sparse. Such information is needed to guide screening and counseling in routine diagnostic settings, where many women first present. Clarifying these links could help clinicians prioritize women at greatest metabolic risk and support early lifestyle intervention before cardiometabolic disease becomes established. This study therefore aimed to examine the association between obesity and metabolic abnormalities among women with PCOS attending a diagnostic centre in Chattogram, Bangladesh and to assess whether abdominal obesity is associated with insulin resistance, hypertriglyceridemia, low HDL-C and impaired fasting glucose.

METHODS & MATERIALS

This cross-sectional analytical study was conducted at Popular Diagnostic Centre Ltd., Chattogram Branch, Bangladesh, from January to December 2021. The study population comprised 150 women with polycystic ovary syndrome diagnosed by the Rotterdam criteria.

Selection Criteria

Inclusion criteria

1. Reproductive-aged women diagnosed with PCOS by the Rotterdam criteria
2. Complete clinical and laboratory data

Exclusion criteria

1. Pregnancy or lactation
2. Known diabetes mellitus
3. Thyroid disease, hyperprolactinemia, Cushing syndrome or congenital adrenal hyperplasia
4. Use of metformin, hormonal contraceptives, corticosteroids, antihypertensive or lipid-lowering drugs

Data Collection Procedure

Eligible women were identified during consultation and informed consent was obtained after the study purpose was explained. Trained personnel collected data using a pre-tested structured questionnaire and a standardized data sheet. Height and weight were measured with participants wearing light clothing and no footwear, using a stadiometer and a calibrated digital scale. Body mass index was calculated as weight in kilograms divided by height in metres squared and categorized using Asian-specific cut-offs. Waist circumference was measured at the midpoint between the lower costal margin and the iliac crest at the end of normal expiration. Blood pressure was recorded in the sitting position after five minutes of rest and the mean of two readings was used. After an overnight fast, venous blood was drawn under aseptic precautions and analyzed in the centre's laboratory for glucose, insulin, triglycerides, HDL-C and LDL-C using standard operating procedures with routine internal quality control. Insulin resistance was estimated by HOMA-IR and a value of 2.5 or above was considered insulin resistance. Metabolic syndrome was defined using the International Diabetes Federation criteria and dyslipidemia, low HDL-C, hypertriglyceridemia and impaired fasting glucose were classified using the thresholds shown in the tables. Confidentiality was maintained by assigning code numbers, removing personal identifiers and restricting data access to the research team. Participants were assured that their data would be used only for research.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation and compared using independent-samples t-test or Mann–Whitney U test and categorical variables as frequency and percentage and compared using chi-square or Fisher's exact test. Binary logistic regression estimated crude and age-adjusted odds ratios with 95% confidence intervals. A p-value below 0.05 was considered significant.

RESULTS

Among 150 women with PCOS (mean age 24.8 ± 5.1 years), 43.3% were obese, 64.7% had abdominal obesity, 52.0% had metabolic syndrome and 80.7% had dyslipidemia. Women with obesity had higher waist circumference, blood pressure, fasting insulin, HOMA-IR and triglycerides, lower HDL-C and a greater prevalence of most metabolic abnormalities, with the exception of impaired fasting glucose. Abdominal obesity was associated with higher odds of insulin resistance, hypertriglyceridemia and low HDL-C.

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Table I: Baseline characteristics of women with PCOS (*n* = 150)

Characteristic	Category	Frequency (n)	Percentage (%)
Age (years)	≤24	78	52.0
	25–29	49	32.7
	≥30	23	15.3
BMI (kg/m^2)	<23	47	31.3
	23–24.9	38	25.3
	≥25	65	43.3
Waist circumference	<80 cm	53	35.3
	≥80 cm	97	64.7
Metabolic syndrome (IDF criteria)	Present	78	52.0
	Absent	72	48.0
Dyslipidemia	Present	121	80.7
	Absent	29	19.3

Table I presents the baseline characteristics of the 150 participants. Most women were aged 24 years or younger (52.0%), followed by those aged 25 to 29 years (32.7%) and 30 years or above (15.3%). By BMI, 47 women (31.3%) were below 23 kg/m^2, 38 (25.3%) were overweight and 65 (43.3%) were obese. A waist circumference of 80 cm or more was recorded in 97 women (64.7%). Metabolic syndrome was present in 78 women (52.0%) and dyslipidemia in 121 (80.7%).

Table II: Anthropometric and biochemical parameters according to BMI-defined obesity status

Parameter	Non-obese (n = 85)	Obese (n = 65)	p-value
Age (years)	24.3 ± 4.9	25.5 ± 5.3	0.152
Waist circumference (cm)	79.2 ± 7.4	93.6 ± 9.8	<0.001
Systolic blood pressure (mmHg)	116.8 ± 11.2	124.5 ± 13.6	<0.001
Diastolic blood pressure (mmHg)	74.9 ± 8.1	78.3 ± 9.2	0.018
Fasting glucose (mmol/L)	5.1 ± 0.6	5.3 ± 0.8	0.082
Fasting insulin (µIU/mL)	9.8 ± 5.2	16.4 ± 9.7	<0.001
HOMA-IR	2.2 ± 1.4	3.9 ± 2.6	<0.001
Triglycerides (mg/dL)	118.4 ± 52.6	156.2 ± 71.3	<0.001
HDL-C (mg/dL)	46.8 ± 10.2	39.5 ± 8.7	<0.001
LDL-C (mg/dL)	108.5 ± 32.4	116.8 ± 35.9	0.142

Table II compares anthropometric and biochemical parameters between non-obese (n = 85) and obese (n = 65) women. Obese women had higher mean waist circumference (93.6 ± 9.8 vs. 79.2 ± 7.4 cm), systolic blood pressure (124.5 ± 13.6 vs. 116.8 ± 11.2 mmHg) and diastolic blood pressure (78.3 ± 9.2 vs. 74.9 ± 8.1 mmHg). Fasting insulin (16.4 ± 9.7 vs. 9.8 ± 5.2 µIU/mL), HOMA-IR (3.9 ± 2.6 vs. 2.2 ± 1.4) and triglycerides (156.2 ± 71.3 vs. 118.4 ± 52.6 mg/dL) were higher, whereas HDL-C was lower (39.5 ± 8.7 vs. 46.8 ± 10.2 mg/dL). These differences were significant ($p < 0.001$, except diastolic blood pressure, $p = 0.018$). Age ($p = 0.152$), fasting glucose ($p = 0.082$) and LDL-C ($p = 0.142$) did not differ significantly.

Table III: Prevalence of metabolic abnormalities according to BMI-defined obesity status

Metabolic abnormality	Non-obese (n = 85), n (%)	Obese (n = 65), n (%)	p-value
Metabolic syndrome (IDF criteria)	32 (37.6)	46 (70.8)	<0.001
Insulin resistance (HOMA-IR ≥2.5)	32 (37.6)	47 (72.3)	<0.001
Dyslipidemia	58 (68.2)	63 (96.9)	<0.001
Low HDL-C (<50 mg/dL)	49 (57.6)	51 (78.5)	0.007
Hypertriglyceridemia (≥150 mg/dL)	22 (25.9)	38 (58.5)	<0.001
Impaired fasting glucose (≥5.6 mmol/L)	9 (10.6)	14 (21.5)	0.065

Table III shows the prevalence of metabolic abnormalities by obesity status. Metabolic syndrome was present in 70.8% of obese and 37.6% of non-obese women ($p < 0.001$) and insulin resistance in 72.3% and 37.6% ($p < 0.001$). Dyslipidemia affected 96.9% and 68.2% ($p < 0.001$), low HDL-C 78.5% and 57.6% ($p = 0.007$) and hypertriglyceridemia 58.5% and 25.9% ($p < 0.001$). Impaired fasting glucose was found in 21.5% and 10.6%, a difference that was not significant ($p = 0.065$).

Table IV: Association between abdominal obesity and individual metabolic abnormalities among women with PCOS (*n* = 150)

Metabolic outcome	Crude OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Insulin resistance (HOMA-IR ≥2.5)	4.32 (2.18–8.56)	<0.001	3.86 (1.94–7.68)	<0.001
Hypertriglyceridemia (≥150 mg/dL)	4.03 (2.01–8.08)	<0.001	3.42 (1.68–6.96)	<0.001
Low HDL-C (<50 mg/dL)	2.68 (1.32–5.44)	0.006	2.71 (1.32–5.56)	0.006
Impaired fasting glucose (≥5.6 mmol/L)	2.32 (0.94–5.72)	0.068	2.14 (0.86–5.32)	0.103

Table IV presents the association between abdominal obesity and individual metabolic abnormalities. In crude analyses, abdominal obesity was associated with higher odds of insulin resistance (OR 4.32, 95% CI 2.18–8.56), hypertriglyceridemia (OR 4.03, 95% CI 2.01–8.08) and low HDL-C (OR 2.68, 95% CI 1.32–5.44). After adjustment for age category, the associations remained significant for insulin resistance (adjusted OR 3.86, 95% CI 1.94–7.68), hypertriglyceridemia (adjusted OR 3.42, 95% CI 1.68–6.96) and low HDL-C (adjusted OR 2.71, 95% CI 1.32–5.56). Impaired fasting glucose showed no significant association in crude (OR 2.32, 95% CI 0.94–5.72; p = 0.068) or adjusted analysis (adjusted OR 2.14, 95% CI 0.86–5.32; p = 0.103). Model fit was acceptable for all outcomes (Hosmer–Lemeshow p = 0.587 to 0.741).

DISCUSSION

In this cross-sectional study of 150 women with PCOS, 43.3% were obese by Asian-specific BMI criteria, 64.7% had abdominal obesity, 52.0% met the IDF criteria for metabolic syndrome and 80.7% had dyslipidemia. The metabolic syndrome prevalence exceeds the pooled estimate of 26.3% reported by Hallajzadeh et al., although their estimates ranged from 7.1% to 37.5% according to the diagnostic criteria applied [12]. It is closer to the figure of 42.9% reported by Ishrat and Hussain among infertile Bangladeshi women with PCOS, who also found dyslipidemia in 93.7% and central obesity in 80.2% [15]. The somewhat lower frequency of dyslipidemia and central obesity in our sample may reflect differences in clinical setting and case mix.

Women with obesity in the present study had higher waist circumference, blood pressure, fasting insulin and HOMA-IR and metabolic syndrome was more frequent than in non-obese women (70.8% vs. 37.6%). Kamrul-Hasan and Aalpona similarly observed in 523 Bangladeshi women with PCOS that metabolic syndrome was far more frequent in the obese group (61.3% vs. 14.8%), although they defined obesity at a BMI of 23 kg/m^2 or above [16]. The smaller contrast in our data is probably explained by our non-obese group, which included 38 women with a BMI of 23 to 24.9 kg/m^2. Ishrat and Hossain found that obesity was strongly associated with waist circumference and fasting insulin and that a BMI of 23 kg/m^2 or above was associated with insulin resistance and metabolic syndrome in infertile women with PCOS [17]. Sachdeva et al. likewise reported that obese women with PCOS carried a higher risk of hypertension, insulin resistance and metabolic syndrome [18].

Lipid abnormalities showed a clear gradient, with dyslipidemia in 96.9% of obese and 68.2% of non-obese women, low HDL-C in 78.5% and 57.6% and hypertriglyceridemia in 58.5% and 25.9%. Overall, low HDL-C was present in two thirds of our participants, which is lower than the 90.5% reported by Ishrat and Hussain, who identified it as the commonest lipid abnormality [15]. Insulin resistance by HOMA-IR affected 52.7% of our cohort, compared with 27.8% in the same Bangladeshi series [15], a difference that may partly reflect differences in sample characteristics and cut-off values.

Metabolic abnormalities were also common among women without obesity, of whom 37.6% had insulin resistance and 68.2% had dyslipidemia. This agrees with Cassar et al., whose meta-analysis of euglycemic clamp studies showed insulin sensitivity about 27% lower in PCOS independent of BMI, with a higher BMI adding a further reduction and with Stepto et al., who documented a high prevalence of insulin resistance in both lean and overweight women with PCOS [9,10]. Zhu et al. also concluded that non-obese women with PCOS experience metabolic disturbances and carry a risk of long-term metabolic complications[19] and Kamrul-Hasan and Aalpona recommended screening all women with PCOS irrespective of BMI category [16].

Abdominal obesity was associated with roughly fourfold crude odds of insulin resistance (OR 4.32) and hypertriglyceridemia (OR 4.03) and with 2.68-fold odds of low HDL-C and these associations persisted after adjustment for age. Glueck and Goldenberg explain that obesity increases insulin resistance and compensatory hyperinsulinemia and raises inflammatory adipokines, which in turn aggravate insulin resistance [8]. Rojas et al. describe a self-reinforcing cycle among hyperinsulinemia, hyperandrogenemia and insulin resistance, with chronic inflammation present in both lean and obese women [5].

Impaired fasting glucose did not differ significantly between obese and non-obese women (21.5% vs. 10.6%) and was not significantly associated with abdominal obesity. Kamrul-Hasan and Aalpona recorded prediabetes in 24.4% of obese and 13.1% of lean women [16], proportions close to ours. Ollila et al. found that the risk of type 2 diabetes was elevated in overweight and obese women with PCOS but not in those of normal weight [14] and Kakoly et al. emphasized the relevance of ethnicity and obesity and the need for accurate diagnostic methods for diabetes [13], so a non-significant fasting glucose finding should not be read as absence of glycemic risk. Given that Kakoly et al. described an established link between PCOS and metabolic health [20] and that Wekker et al. showed an increased long-term cardiometabolic risk in PCOS [21], early metabolic evaluation and weight management appear justified for all women with PCOS.

LIMITATIONS & RECOMMENDATIONS

This study has limitations. Its cross-sectional design precludes causal inference, the single-centre setting and modest sample size limit generalizability and adjustment was restricted to age. Insulin resistance was estimated using HOMA-IR and dysglycemia was assessed by fasting glucose rather than an oral glucose tolerance test. Future multicentre longitudinal studies with larger samples androgen profiling and body composition measures are recommended. Clinically, routine metabolic screening and structured weight management should be offered to women with PCOS regardless of BMI.

CONCLUSION

Obesity was significantly associated with adverse metabolic profiles among women with PCOS. Women with a BMI of 25 kg/m^2 or above had higher blood pressure, fasting insulin, HOMA-IR and triglycerides, lower HDL-C and a greater prevalence of metabolic syndrome, insulin resistance and dyslipidemia. Abdominal obesity remained associated with insulin resistance, hypertriglyceridemia and low HDL-C after age adjustment. Metabolic abnormalities were common in non-obese women. Metabolic screening and weight management should be integrated into the care of all women with PCOS.

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